A Dissertation on

ECHOCARDIOGRAPHIC EVALUATION OF LEFT VENTRICULAR SYSTOLIC AND DIASTOLIC DYSFUNCTION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

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CERTIFICATE

This is to certify that this dissertation entitled "**ECHOCARDIOGRAPHIC EVALUATION OF LEFT VENTRICULAR SYSTOLIC AND DIASTOLIC DYSFUNCTION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION**" submitted by Dr. STALIN ROY .J, to the Tamil Nadu Dr.MGR Medical University is in partial fulfilment of the requirement for the award of M.D. DEGREE (BRANCH -1) and is a bonafide research work carried out by him under direct supervision and guidance.

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ABBREVIATIONS AND ACRONYMS

AMI	Acute Myocardial Infarction
ASMI	Antero Septal Myocardial Infarction
DD	Diastolic Dysfunction
DT	Deceleration Time
LVEF	Left Ventricular Ejection Fraction
LVEDD	Left Ventricular End Diastolic Diameter
LVESD	Left Ventricular End Systolic Diameter
IVCT	Isovolumic Contraction Time
IPWMI	Inferoposterior Wall Myocardial Infarction
IWMI	Inferior Wall Myocardial Infarction
LA	Left Atrium
LVET	Left Ventricular Ejection Time
MI	Myocardial Infarction
MPI	Myocardial Performance Index
RV	Right Ventricle
RVMI	Right Ventricular Myocardial Infarciton
RWMA	Regional Wall Motion Analysis
RWMI	Regional Wall Motion Scoring Index
TDI	Tissue Doppler Imaging
WMSI	Wall Motion Scoring Index

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INTRODUCTION

Acute Myocardial Infarction is one of the leading causes of death among men and women worldwide. Most of the early deaths are due to Ventricular Arrhythmias. These Arrhythmias are responsible for the sudden deaths associated with Myocardial Infarction. The late mortality associated with Myocardial Infarction is typically due to left ventricular dysfunction and its complications. Residual Left Ventricular function after Myocardial Infarction is an important prognostic marker.

Left Ventricular dysfunction can be systolic, diastolic or both. Echocardiography is the most widely used and readily available, non-invasive tool in the arsenal of cardiologist for evaluating the left ventricular function. Echocardiographic evaluation of Left Ventricular function is an integral part of evaluation of a patient with Acute Myocardial Infarction. Two – Dimensional echocardiography is useful for the assessment of systolic function, and Doppler Echocardiography is well suited for studies of diastolic function.

An acute Trans-mural Myocardial Infarction causes a loss of contractile fibres which reduces systolic function. Parallel to the effect on systolic function, a myocardial infarction also impacts diastolic function, as evidenced by the raise in left ventricular end diastolic pressure.

This study is performed to estimate the prevalence of left ventricular systolic and diastolic dysfunction using various Echocardiographic indices in

patients with Acute ST elevation Myocardial Infarction and to find out its significance in determining early in-hospital morbidity, especially early Congestive Heart Failure in such patients.

AIMS AND OBJECTIVES OF THE STUDY

1) To assess the prevalence of Left Ventricular Systolic and Diastolic dysfunction in patients with Acute Myocardial Infarction.

2) To study the association between Left Ventricular Systolic, Diastolic dysfunction and the variables such as Age, Sex, Smoking, Diabetes, Hypertension, Killip Class, Type of Myocardial Infarction.

3) To assess the relationship between the echocardiographic indices of systolic and diastolic function and the development of early in-hospital congestive heart failure (as defined by Killip Class $\geq$ II).

REVIEW OF LITERATURE

Acute ST Elevation Myocardial Infarction – an overview

Despite the advances made in the diagnosis and management of ST Elevation Myocardial Infarction (STEMI), it continues to be a major public health problem in the industrialized world as well as in developing countries like India. [1] It has been estimated that the number of years of life lost because of an AMI is 15 years. The burden of Myocardial Infarction in developing countries is approaching those now afflicting developed countries. The scarcity of available resources to treat ST Elevation Myocardial Infarction (STEMI) in developing countries mandate major efforts on an international level to strengthen primary prevention programs.[2]

Mortality from STEMI has declined steadily over the past few decades.[3] This drop in mortality appears to result from a fall in the incidence of STEMI which is replaced in part by an increase in the rate of Unstable Angina(UA)/non–ST-segment elevation Myocardial Infarction (NSTEMI)[4] and a fall in the case fatality rate of STEMI patients.[5] Although reperfusion has made important progress in lowering mortality, many patients with acute MI are not eligible for this therapy and face in-hospital death rates of 10% to 20%.[6]

Historical phases in the evolution of coronary care

There have been several phases in the management of patients with STEMI. These phases have contributed to the gradual decline in mortality from STEMI.[7] In the first half of the 20th century, management of STEMI focussed on a detailed recording of physical and laboratory findings, with little active treatment for the infarction. This phase is known as the "clinical observation phase" of coronary care. The "coronary care unit phase" began in the mid-1960s and included detailed analysis and vigorous management of cardiac arrhythmias. The "high-technology phase" began with the introduction of the pulmonary artery balloon flotation catheter. It helped in the bedside hemodynamic monitoring and more precise hemodynamic management of STEMI patients. The modern "reperfusion era" of coronary care heralded the introduction of intracoronary and then intravenous fibrinolysis, increased use of aspirin, and the development of primary percutaneous coronary intervention. Contemporary care of patients with STEMI has entered an evidence-based coronary care phase where we are increasingly using standard guidelines and performance measures for clinical practice.[8]

Acute myocardial infarction (AMI) results in local myocyte damage that leads to systolic and diastolic dysfunction. Following a myocardial infarction various physiological and pathophysiological process are set in motion. Some of them are left ventricular (LV) remodelling, local and systemic neurohormonal

activation, and vascular dysfunction. LV systolic dysfunction, its pathophysiology and prognosis after AMI have been extensively researched for several decades. Either clinical or radiographic evidence of heart failure has been found to be a powerful predictor of outcome in patients after AMI, in addition to depressed systolic function.[9] Pulmonary congestion after infarction has been attributed to raised LV filling pressures but it may be seen after what appears to be only minor myocardial damage.[10] The pathophysiological basis for raised filling pressures is incompletely understood but may involve impaired active relaxation of the myocardium and increased LV chamber stiffness. These abnormalities constitute what is known as diastolic dysfunction.

Definition and Clinical evaluation of patients with myocardial infarction

Acute Myocardial infarction (AMI) can be defined from a number of different perspectives related to clinical, electrocardiographic (ECG), biochemical and pathologic characteristics. The gold standard for diagnosing myocardial infarction has been the World Health Organization definition, [11] which requires any 2 of 3 criteria:

1. Ischemic symptoms,
2. Elevated creatine kinase-MB levels, and
3. Electrocardiographic changes.

Recently, a new definition that for the first time which includes elevated troponin levels have been published by the American College of Cardiology and the European Society of Cardiology published.

Clinical Features

The classic symptoms of MI are intense, oppressive, durable, excruciating chest pressure, with an impending sense of doom and radiation of the pain to the left arm. However, the other symptoms of chest heaviness or burning, radiation to the jaw, neck, shoulder, back, or both arms may be encountered. The discomfort is not affected by moving the muscles of the region where the discomfort is located, nor is it worsened by respiratory movements and not positional in nature. The discomfort associated with acute MI usually lasts at least 20 min, but may be shorter in duration. The pain is usually sustained, but can be stuttering. Nausea and Vomiting are frequently encountered in inferior wall myocardial infarction. Another typical finding is profuse diaphoresis. On the whole, the classical presentation is experiencing a unique, discrete, painful event that has induced fear. However, exceptions to the classical presentation are common and are more challenging. It is imperative to ask whether there were premonitory signs of chest discomfort in the preceding week or two. Other associated risk factors, such as smoking, elevated cholesterol, diabetes, hypertension, and family history, when present, give us a supportive piece that helps to put the acute history into context. Dyspnea, when present denotes

incipient congestive heart failure or, alternatively, is an outgrowth of the patient's anxiety. Palpitations or syncope are quite uncommon, but a history of light-headedness or dizziness and presyncope often reflects the underlying vagotonia or bradyarrhythmias seen in inferior wall myocardial infarction. Ventricular tachycardia may produce syncope or an out-of-hospital arrest.

Although most of the patients have symptoms just described, these complaints may go unrecognized or may be erroneously labelled as another disease entity, such as peptic ulcer disease. Myocardial necrosis may also occur without symptoms; it may be detected only by the Electrocardiogram, raised cardiac enzymes or other imaging studies.

Risk stratification of patients with Acute Myocardial Infarction

The physical examination also provides a method for the risk stratification of STEMI patients. The Killip classification (shown in the table) can be used as a method to stratify patients and predict clinical outcomes.[9] The Killip classification originally devised in the 1960's has stood the test of time and has been shown in several studies to accurately predict clinical outcomes following Acute Myocardial Infarction. With modern therapy, the mortality of those in cardiogenic shock has improved from 83% (at the time the study was done) to approximately 60% now.

Killip Classification for Patients with STEMI[9]		
Killip Class		**Hospital Mortality (%)**
I	**No congestive heart failure**	6
II	**Mild congestive heart failure, rales, S_3, congestion on chest radiograph**	17
III	**Pulmonary edema**	38
IV	**Cardiogenic shock**	81*

* Has improved to approximately 60% with current therapy.

Clinical classification of Myocardial Infarction [12]

Clinical Classification of Different Types of Myocardial Infarction[12]	
Type 1	Spontaneous myocardial infarction related to ischaemia due to a primary coronary event such as plaque erosion and/or rupture, fissuring, or dissection
Type 2	Myocardial infarction secondary to ischaemia due to either increased oxygen demand or decreased supply, e.g. coronary artery spasm, coronary embolism, anaemia, arrhythmias, hypertension, or hypotension
Type 3	Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischaemia, accompanied by presumably new STelevation, or new LBBB, or evidence of fresh thrombus in a coronary artery by angiography and/or at autopsy, but death occurring before blood samples could be obtained, or at a time before the appearance of cardiac biomarkers in the blood
Type 4a	Myocardial infarction associated with PCI
Type 4b	Myocardial infarction associated with stent thrombosis as documented by angiography or at autopsy
Type 5	Myocardial infarction associated with CABG

Table adapted from Thygeson et al[12]

The Joint Task Force of the European Society of Cardiology, American College of Cardiology Foundation, the American Heart Association, and the World Health Federation (ESC/ACCF/AHA/WHF) in 2007, has proposed the above clinical classification of myocardial infarction.

Echocardiography in Patients with Acute Myocardial Infarction

Echocardiography has several important roles, in patients with AMI:

(1) Diagnosis and exclusion of acute MI in patients with prolonged chest pain and non-diagnostic electrocardiographic findings;

(2) Estimation of the amount of myocardium at risk and final infarct size after reperfusion therapy;

(3) Evaluation of patients with unstable hemodynamic findings and detection of infarct complications;

(4) Evaluation of myocardial viability; and

(5) Risk stratification.

Two-dimensional echocardiographic imaging is helpful in assessing reperfused myocardial segments or infarct expansion in patients with STEMI. When the segments are persistently akinetic, it does not always indicate failed

reperfusion. If the myocardium remains akinetic while being viable, dobutamine stress echocardiography is helpful to demonstrate its viability.[13,14]

Detection of Mechanical Complications of Acute Myocardial Infarction

The mechanical complications of Myocardial Infarction can be life-threatening. Hence it is imperative that reliable and timely identification is critical for optimal management. In a patient with suspected mechanical complication or with unstable hemodynamics, Two-dimensional (2D) and Doppler echocardiography with colour flow imaging is generally the first imaging modality used. TEE (Transesophageal Echocardiogram) is of immense utility for patients in whom precordial echocardiography is not possible for various reasons. TEE can be used under the most difficult clinical situations, including in the critical care unit, in intubated patients and postoperative patients, and even during cardiopulmonary resuscitation.[15] Mechanical complication should be suspected when a critically ill or hemodynamically unstable patient has normal systolic function.

Evaluation of Systolic and Diastolic Function

Systolic Functional Parameters

The systolic parameters measured by Echocardiography that are used as a marker for left ventricular systolic function of the heart are Left Ventricular Ejection Fraction (LVEF), stroke volume and cardiac index, systolic tissue

velocity of the mitral annulus, fractional shortening, strain, and regional wall motion analysis.

Left Ventricular Ejection Fraction

The most commonly used and universally accepted expression of global LV function is Left Ventricular Ejection Fraction (LVEF). Although LVEF has many limitations, including operator dependency, it is a strong predictor of clinical outcome in many cardiac conditions. LVEF is also used to select optimal management strategies. In clinical practice, LVEF is usually determined by visual assessment of two-dimensional echocardiographic images of the left ventricle. This method is reasonably reliable when it is performed by an experienced echocardiographer but varies widely among readers. Hence it is advisable that, LVEF should be measured more objectively whenever possible, using volumetric measurements as described by the following equation:

$$\boldsymbol{LVEF} = \frac{(\boldsymbol{LVEDV} - \boldsymbol{LVESV})}{\boldsymbol{LVEDV}}$$

where LVEDV and LVESV are LV end-diastolic volume and end-systolic volume, respectively.

M-mode or two-dimensional echocardiography are used to measure LV dimensions and LVEF can also be calculated from these values. The following formula is used to calculate LVEF from the M-mode or two-dimensional

echocardiographic measurement of LV dimensions from the mid-ventricular level:

$$\boldsymbol{LVEF} = \frac{(\boldsymbol{LVEDD^2} - \boldsymbol{LVESD^2})}{\boldsymbol{LVEDD^2}}$$

Where LVEDD and LVESD are end-diastolic diameter and end-systolic diameter, respectively.

This equation is actually calculates the percentage change in LV area, or fractional shortening of the LV short axis, which equals LVEF if the apical long-axis dimension remains the same from diastolic phase to systolic contraction. Since the apical long axis normally shortens 10% to 15% with systole, an apical correction factor is added on the basis of the contractility of the apex: 5% to 7% for normal to hyperdynamic apical contraction, 3% for hypokinetic contraction, and 0% for akinetic apex.

Three-dimensional (3D) echocardiography is likely to become the standard method to calculate the LVEF, because it can provide LV end-diastolic and end-systolic volumes closer to those measured by Cardiac Magnetic Resonance (CMR). The synchronicity of LV regional contraction can also be assessed by 3D Echocardiography, as it can also provide regional LV volume as well as the timing of the smallest volume of each region.

Fractional Shortening

Fractional shortening (FS) is the percentage change in LV dimensions with each LV contraction. This systolic function parameter is now rarely used for diagnosis or clinical decision making.

$$\boldsymbol{FS} = \frac{(\boldsymbol{LVEDD} - \boldsymbol{LVESD})}{\boldsymbol{LVEDD}}$$

Stroke Volume

Stroke volume (SV) can be measured as the difference between LVEDV and LVESV obtained by the Simpson method or three-dimensional echocardiography. The difference will be equal to systolic volume across the LVOT (Left Ventricular Outflow Tract) if there is no valvular regurgitation. When there is mitral regurgitation (MR), regurgitant volume needs to be subtracted to obtain Stroke Volume across LVOT. The product of LVOT area and LVOT time-velocity integral also give the LVOT Stroke Volume.

Systolic Velocity of Myocardial Tissue or Mitral Annulus

Tissue Doppler Echocardiography is used to measure the systolic component of the mitral annulus. There is good correlation between LVEF and Systolic Velocity of Myocardial Tissue and is a good predictor of outcome in many cardiac disorders.[16]

Regional Wall Motion Scoring Index (RWMI)

Regional wall motion analysis is the most commonly used echocardiographic parameter to evaluate coronary artery disease. It is one of the most common uses of echocardiography.

From the parasternal, apical, and sometimes subcostal imaging windows, two-dimensional echocardiography can visualize all LV wall segments. For purposes of regional wall motion analysis, the ASE has recommended a 16-segment model or, optionally, a 17-segment model with an addition of the apical cap. The following numerical score is assigned to each wall segment on the basis of its contractile function as assessed visually:

1 = normal (>40% thickening with systole);

2 = hypokinesis (10% to 40% thickening);

3 = severe hypokinesis to akinesis (<10% thickening);

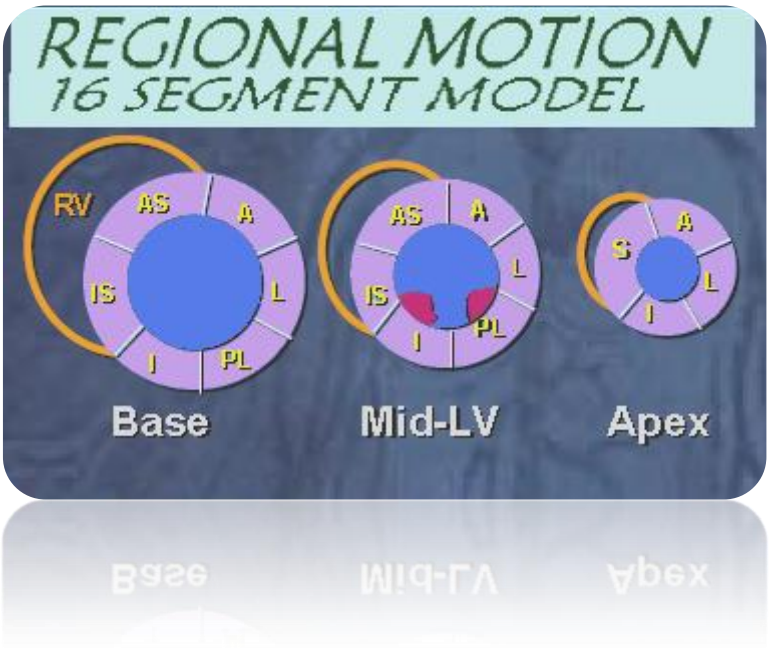
4 = dyskinesis; and

5 = aneurysm.

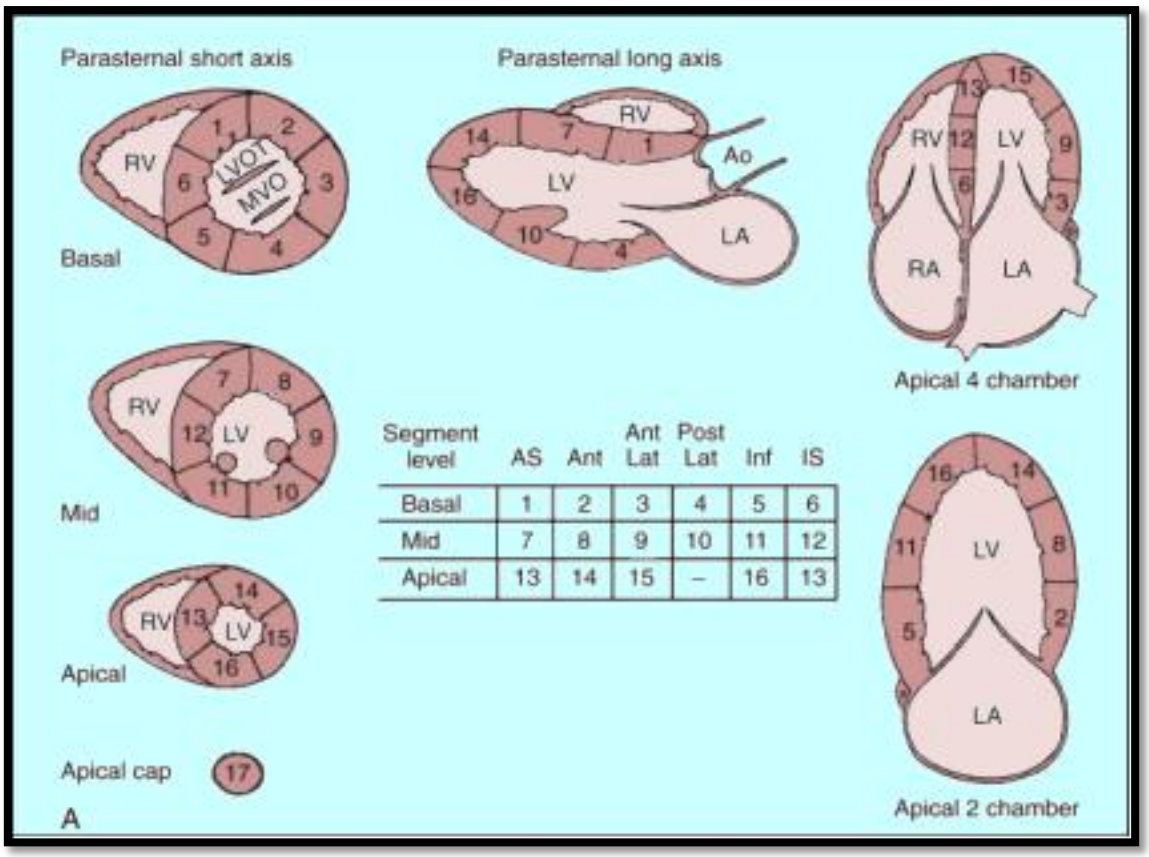
On the basis of this wall motion analysis scheme, a wall motion score index (WMSI) is calculated to semiquantitate the extent of regional wall motion abnormalities:

$$RWMI/WMSI = \frac{Sum\ of\ wall\ motion\ scores}{Number\ of\ segments\ visualised}$$

A normal left ventricle has a WMSI of 1, and the index increases as wall motion abnormalities become more severe. When two-dimensional echocardiography was performed simultaneously with sestamibi SPECT in patients with acute ST-segment MI (STEMI), the overall correlation between the WMSI and the perfusion defect was good. Patients with WMSI higher than 1.7 tend to have a perfusion defect greater than 20%. The correlation is usually better for patients with an anterior wall MI than for those with an inferior or lateral wall MI with a smaller infarct size. A small area of subendocardial ischemia may not demonstrate wall motion abnormality, but contrast echocardiography can demonstrate a rim of subendocardial perfusion defect.

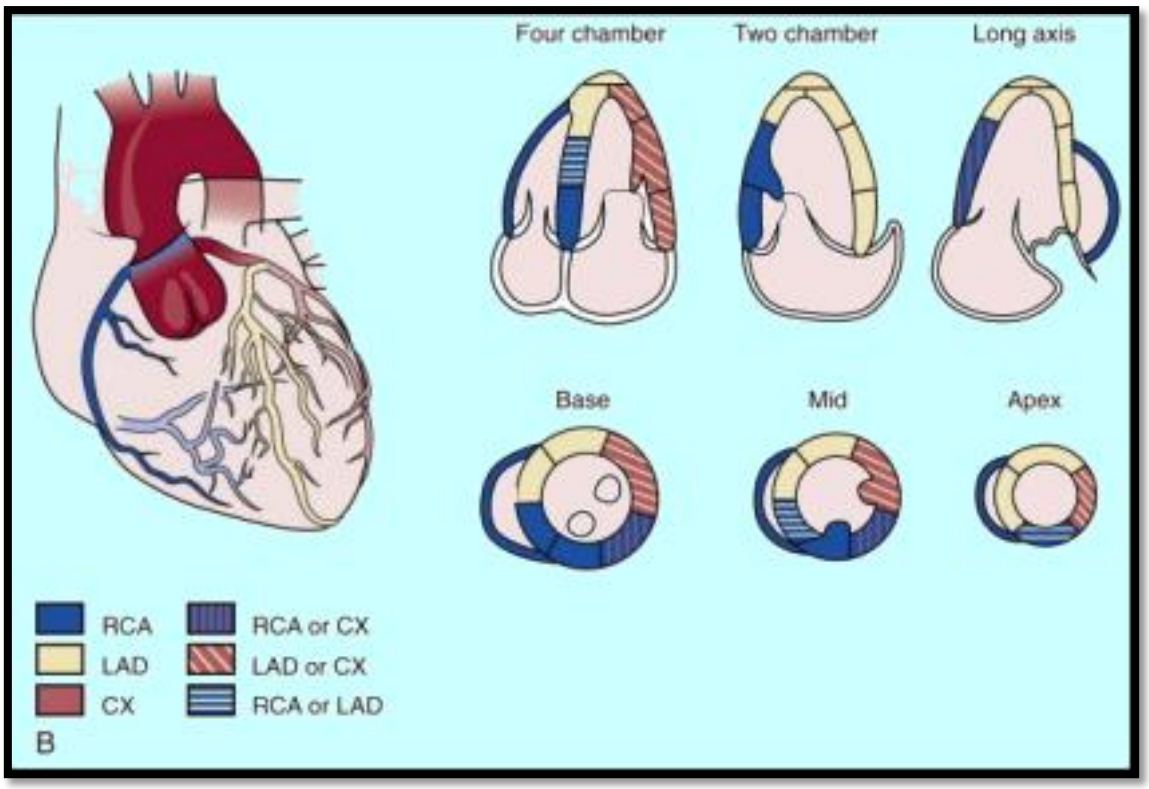


Picture depicting the 16 segmental model for wall motion scoring



Segment level	AS	Ant	Ant Lat	Post Lat	Inf	IS
Basal	1	2	3	4	5	6
Mid	7	8	9	10	11	12
Apical	13	14	15	–	16	13

Echocardiography is helpful in the evaluation of chest pain, especially during active chest pain. The absence of LV wall motion abnormalities during chest pain usually but not always excludes myocardial ischemia or infarction, and the presence of regional wall motion abnormalities has a high sensitivity for detection of myocardial ischemia or infarction, although it is not specific. Myocardial contrast perfusion imaging provides incremental diagnostic value for patients with chest pain, with excellent concordance with gated SPECT (77% to 84%).[17] However, routine use of echocardiography in this setting requires availability of appropriately trained personnel to perform echocardiography and to interpret its findings.



Wall motion analysis can also be performed more objectively and conveniently by speckle tracking strain imaging. Moreover, Ishii and colleagues[18] elegantly demonstrated that diastolic relaxation of the ischemic myocardial segment remains abnormal long after resolution of the regional wall motion abnormality, and the diastolic relaxation abnormality can be detected by strain imaging.

Assessment of Diastolic Function

Thorough evaluation of diastolic function is essential, while assessing the cardiac function of a patient because about half of patients with heart failure have preserved LVEF. Echocardiography has become the preferred non-invasive method to evaluate diastolic function and to estimate left ventricular

filling pressures. M-mode, two-dimensional, and Doppler (blood flow, tissue, and colour) echocardiography are all helpful in evaluating diastolic function. Recently, the American Society of Echocardiography (ASE) and the European Association of Echocardiography (EAE) published a guideline for assessment of diastolic function by echocardiography.[19] The following steps will ensure comprehensive assessment of diastolic function and the identification of heart failure related to diastolic dysfunction:

	ASE and the European Association of Echocardiography (EAE) guideline for assessment of diastolic function by echocardiography
1	Look for M-mode and two-dimensional echocardiographic evidence of diastolic dysfunction. Abnormal myocardial relaxation, an integral part of diastolic dysfunction, decreases the slope (in M-mode) and mitral annulus motion of early diastolic filling and increases LA size. LV wall thicknesses are usually but not necessarily increased.
2	Mitral inflow velocities reflect the transmitral pressure gradient, which is usually characteristic of various stages of diastolic dysfunction. Assessment of ventricular compliance is possible from the configuration (velocities and flow duration) of mitral inflow velocities. Pulmonary vein flow velocities are also helpful.
3	Myocardial relaxation by TDI can be evaluated. Mitral annulus velocity (e') during early diastole correlates reasonably well with the status of myocardial relaxation (tau).
4	Mitral inflow velocities (E and A), e', mitral inflow propagation velocity, and their combination can estimate LV diastolic filling pressure at rest and with exercise.
5	These steps allow diagnosis of diastolic heart failure and separation of myocardial diastolic heart failure from pericardial diastolic heart failure.

LV diastolic filling consists of a series of events that are affected by numerous factors, including myocardial relaxation, compliance, cardiac rhythm, and pericardial compliance. Normal diastolic function ensures adequate filling of the ventricles during rest and exercise without an abnormal increase in diastolic pressure or pulmonary venous congestion. The initial diastolic event is myocardial relaxation,[20] An active energy-dependent process that causes LV pressure to decrease rapidly after the end of contraction. When LV pressure falls below LA pressure, the mitral valve opens, and rapid early diastolic filling begins. Under normal circumstances, a major determinant of the driving force of early diastolic filling is the elastic recoil caused by normal relaxation of the left ventricle. Normally, 75% to 80% of LV filling occurs during this phase. During early diastolic filling, LV pressure continues to decrease until completion of myocardial relaxation (normally about 100 milliseconds) before rising after reaching minimal pressure; this loss of positive driving force results in the deceleration of mitral inflow. Later, atrial contraction produces a positive transmitral pressure gradient and inflow, accounting for 20% to 25% of LV filling in normal subjects. The proportion of LV filling during the early and late diastolic phases depends on elastic recoil (suction), rate of myocardial relaxation, chamber compliance, LA pressure, and heart rate. The LV filling pattern is the result of the transmitral pressure gradient produced by these various factors.

The transmitral pressure gradient or the relationship between LA and LV pressures is accurately reflected by mitral inflow Doppler velocities.[20] Diastolic filling is usually classified initially on the basis of the peak mitral flow velocity of the early rapid filling wave (E), peak velocity of the late filling wave caused by atrial contraction (A), E/A ratio, and deceleration time (DT), which is the time interval for the peak E velocity to reach zero baseline.

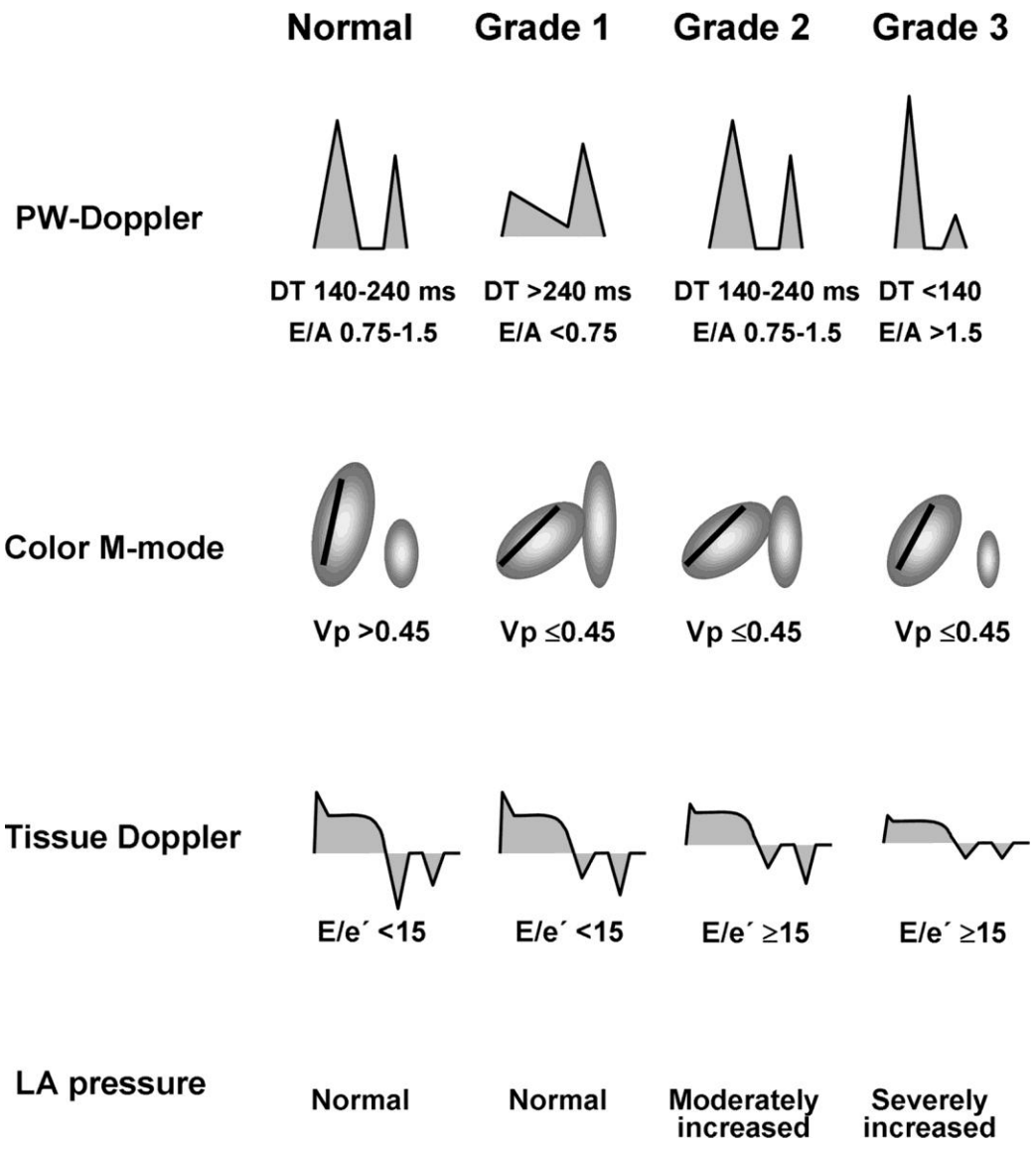
With myocardial relaxation, the LV cavity elongates, expands laterally, and rotates. The longitudinal motion of the mitral annulus has been shown to correlate with the rate of myocardial relaxation. The velocity of the mitral annulus can be recorded by TDI, which has become an essential part of evaluation of diastolic function by echocardiography.[21] Radial and circumferential function can also be assessed with speckle tracking strain imaging.[22]

Comprehensive assessment of diastolic filling and estimation of filling pressures by echocardiography require Tissue Doppler Imaging (TDI), pulmonary vein Doppler, hepatic vein Doppler, and colour M-mode of mitral inflow for propagation velocity—sometimes with an alteration in a loading condition. The Valsalva maneuver is used most frequently to decrease venous return by increasing intrathoracic pressure.[23]

Grading of Diastolic Dysfunction (or Diastolic Filling Pattern)

The grading of the diastolic filling pattern (or diastolic dysfunction) is based on several parameters.[24] In most (if not all) cardiac diseases, the initial diastolic abnormality is impaired relaxation. With further progression of disease and a mild to moderate increase in LA pressure, the mitral inflow velocity pattern appears similar to a normal filling pattern (pseudonormalized). With further decrease in LV compliance and increase in LA pressure, diastolic filling becomes restrictive. Most patients with restrictive filling are symptomatic and have a poor prognosis unless the restrictive filling can be reversed by treatment. However, restrictive filling may be irreversible and represent the end stage of diastolic heart failure. Therefore, diastolic dysfunction can be graded according to the diastolic filling pattern.[19]

- Grade 1 (mild dysfunction) - impaired relaxation with normal filling pressure
- Grade 2 (moderate dysfunction) - pseudonormalized mitral inflow pattern
- Grade 3 (severe reversible dysfunction) - reversible restrictive (high filling pressure)
- Grade 4 (severe irreversible dysfunction) - irreversible restrictive (high filling pressure)



Grading of diastolic dysfunction by echocardiography[38]

A grade 1 diastolic filling pattern usually implies a normal filling pressure despite a background of impaired myocardial relaxation. However, in patients with a marked relaxation abnormality, as in Hypertrophic Cardiomyopathy, the

filling pressure can still be elevated with grade 1 mitral inflow velocity pattern (E/A ratio <1.0 and DT >240 milliseconds). Because the reversibility of restrictive filling usually cannot be assessed at one clinical setting, grade 4 dysfunction was not used in the standard recommendations.[19]

Clinical Applications of Diastolic Function Assessment

Assessment of diastolic function echocardiographically has the following clinical applications and should be an integral part of an echocardiography examination.

1. Estimation of filling pressures at rest and with exercise:-

In patients with reduced LV systolic function (LVEF <35%), mitral inflow E/A ratio of 1.5 or higher and DT of 140 milliseconds or higher indicate increased filling pressures. However, these parameters do not have a good correlation with filling pressure in patients with normal LVEF and diastolic heart failure. For all degrees of LVEF, E/e′ is the best parameter to estimate filling pressure; pulmonary capillary wedge pressure (PCWP) is 20 mm Hg or more if E/e′ is 15 or higher, and PCWP is normal if E/e′ is less than 8.[19] When E/e′ is 8 or higher but less than 15, pulmonary vein flow duration and the Valsalva maneuver can help estimate PCWP. In an important subset of patients with diastolic dysfunction, PCWP is normal at rest but increases only with

exertion, causing exertional dyspnea. It is feasible and reliable to estimate PCWP with exercise by recording mitral inflow and annulus velocity. In a normal population with normal diastolic function, filling pressure rarely increases with exercise. Diastolic dysfunction (or impaired myocardial relaxation) is usually a prerequisite for development of exercise-induced high filling pressure. These patients increase cardiac output at the expense of increased filling pressure. In this situation, mitral E velocity increases while annulus Ea velocity does not increase as much or at all, resulting in an increase in E/e′ ratio. E/e′ correlates well with simultaneously measured PCWP with exercise as well as during resting stage, and a ratio higher than 15 indicates PCWP greater than 20 mm Hg with exercise.[25]

2. Diagnosis of cardiomyopathies, and constrictive pericarditis:-

Knowledge of the diastolic filling pattern and filling pressures allows the detection of cardiac diseases that are frequently missed or not suspected clinically, especially when the LVEF is normal. Patients with diastolic heart failure and normal LVEF have a large LA volume and evidence of impaired relaxation as well as increased filling pressure. There are several reports that TDI of myocardial relaxation can diagnose various forms of cardiomyopathy (HCM, Fabry disease, and amyloidosis) even before frank phenotypic manifestation.[26] The detection of constrictive pericarditis has been made much easier with the use of echocardiographic diastolic parameters and TDI.[27]

3. Prognosis:-

Diastolic echocardiographic parameters, E, E/A, DT, E/e′, and LA volume, have been found to be powerful prognostic indicators for various conditions.[28,29] Even in asymptomatic patients, the presence of diastolic dysfunction portends a poor clinical outcome.

Although diastolic filling is affected by various factors, the direction of its change or progression is predictable in patients with known heart disease. Therefore, assessment of the diastolic filling pattern allows LV filling pressures and LV compliance and relaxation to be estimated and understood so that optimal treatment strategies can be offered to symptomatic patients with diastolic dysfunction.

Evaluation of Cardiac Function by Cardiac Time Intervals

Cardiac time intervals are valuable tools which gives us a clear insight into systolic and diastolic function of the heart. Initially the Isovolumic contraction time (IVCT) duration and the preejection period (PEP) were analysed extensively as a measure of cardiac systolic function. The LV Stroke Volume was derived from the left ventricular ejection time. Myocardial dysfunction prolongs PEP and shortens LVET. However these intervals are also influenced by many other hemodynamic and electrical variables other than systolic dysfunction. An index called systolic time interval (PEP/LVET) was

derived by Weissler and colleagues, which was less heart rate dependent as a measure of LV systolic function. Because LV dysfunction also affects IVRT is also affected, Mancini and colleagues incorporated IVRT into an index called the isovolumic index, derived as (IVCT + IVRT)/LVET. The sum of IVCT and IVRT was measured by subtracting LVET from the peak of the R wave on the electrocardiogram to the onset of mitral valve opening. The isovolumic index was considered more sensitive for cardiac dysfunction than the systolic time interval because it contains IVRT as well as IVCT. However, the interval from the R wave peak to the onset of mitral valve opening contains an interval of electromechanical delay, which can be pronounced in patients with left bundle branch block. With the advent of Doppler echocardiography, it has become easier to determine cardiac time intervals more reliably. Tei and colleagues proposed an index of myocardial performance with Doppler echocardiography (IMP or Tei index) that is independent of the electromechanical delay, (IVCT + IVRT)/LVET, and can be used to identify the exact onset of isovolumic contraction.[29]

The time intervals necessary for calculation of IMP are easily obtained with Doppler echocardiography and TDI[30] as well as with M-mode echocardiography.[31] The normal value is 0.39 ± 0.05, and its mean value is 0.59 ± 0.10 in those with dilated cardiomyopathy. The IMP was evaluated for the right ventricle, especially in patients with pulmonary hypertension. When

myocardial relaxation is normal, the opening of the mitral valve is initiated by rapid suction of the left ventricle; hence, the onset of mitral valve opening is close to the onset of early diastolic movement (or velocity) of the mitral annulus.[32] However, if myocardial relaxation is delayed, the mitral valve opens by high LA pressure. Therefore, the onset of diastolic motion of the mitral annulus follows the onset of mitral inflow. The time interval has been correlated with the degree of impairment in myocardial relaxation and LV filling pressure. With worsening of diastolic function, the time interval lengthens.

Echocardiographic indices to assess the prognosis following Acute Myocardial Infarction

Doppler echocardiographic assessment of hemodynamics in the acute setting of AMI provides independent, rapid, feasible, and simple non-invasive method of assessing the prognostic factors. This is particularly true in the subgroup of patients who have evidence of elevated LV filling pressures despite relatively preserved systolic function.

The most important prognostic indicators after Myocardial Infarction are the degree of LV systolic dysfunction, left ventricular end-systolic volume index, ejection fraction, infarct size as peak cardiac enzyme release, infarct location and transmurality, LV volume, LV sphericity, Mitral Regurgitation, diastolic function, frequent ventricular arrhythmias and presence of heart

failure.[33,34,35] Therefore, it is reasonable to predict that patients with a high WMSI have a greater chance for subsequent development of cardiac events. Most patients with Killip class II-IV heart failure after acute Myocardial Infarction have a WMSI of 1.7 or higher. In addition to the WMSI, restrictive Doppler filling variables derived from mitral inflow velocities correlate well with the incidence of postinfarction heart failure and LV filling pressures.[36,37] The E/e′ ratio, a reliable parameter to estimate PCWP, was found to be a strong predictor for long-term outcome after acute Myocardial Infarction.[36] LA volume, a surrogate for chronic diastolic dysfunction and chronic elevation of LA pressure, was also a strong predictor of outcome.[38] Stress echocardiography is sensitive in detecting residual ischemia, myocardial viability, and multivessel disease soon after Myocardial Infarction[39]. Often, however, patients are unable to exercise adequately soon after an acute Myocardial Infarction, and the myocardium may remain akinetic for a period of days to weeks after successful reperfusion of the occluded coronary artery. Demonstration of viability by augmentation of contractility (with dobutamine echocardiography) or demonstration of perfusion (with contrast echocardiography) predicts functional recovery

In a meta-analysis of 12 prospective clinical trials, of survivors of acute myocardial infarction, Whalley et al[44] studied whether simple, universally available Doppler echocardiographic measurements of left ventricular diastolic

function predict clinical outcome. The final analysis provided important findings. Despite the lack of data on the impact of the precise timing of the Doppler echocardiogram, the different baseline demographics, and the potential influence of discordant postinfarction pharmacotherapies between the 2 groups, two clear conclusions can be drawn. The first conclusion is that a restrictive left ventricular filling pattern, even in the presence of a normal ejection fraction, predicts clinical outcome after infarction. There is a 3-fold increase in risk of death when Restrictive filling is present. A restrictive filling pattern provides incremental prognostic information over and above that of left ventricular volumes and Killip class. The second conclusion that can be drawn is for stratification of patients at increased risk following myocardial infarction, left ventricular filling profiles should be evaluated.

MATERIALS AND METHODS

Individuals who were admitted for acute Myocardial Infarction in the Intensive Coronary Care Unit, Department of Cardiology, Govt. Stanley Hospital from April 2011 to September 2011 were evaluated in this study. Their Left Ventricular Systolic and Diastolic function was assessed by 2D Doppler Echocardiography within 48 hours of admission.

Left Ventricular Ejection Fraction – the most well accepted expression of systolic Left Ventricular function is measured with the help of 2D echocardiography.

Regional Wall Motion Abnormalities are also assessed and graded as:

1 *Normal*

2 *Hypokinesia*

3 *Severe Hypokinesia -akinesia*

4 *Dyskinesia*

5 *Aneurysm*

On the basis of this wall motion analysis scheme, a wall motion score index (WMSI) is calculated to semiquantitate the extent of regional wall motion abnormalities:

$$\boldsymbol{RWMI/WMSI} = \frac{\boldsymbol{Sum\ of\ wall\ motion\ scores}}{\boldsymbol{Number\ of\ segments\ visualised}}$$

A normal left ventricle has a WMSI of 1, and the index increases as wall motion abnormalities become more severe. For purposes of regional wall motion analysis, the ASE has recommended a 16-segment model. Segments are visualised from the parasternal, apical, and subcostal imaging windows. The segments are labelled at three levels – Apical, mid-papillary, Basal. The levels are depicted below:

Table showing segmental levels for RWMI scoring			
Segment level	**Basal**	**Mid-papillary**	**Apical**
Antroseptal	1	7	13
Anterior	2	8	14
Anterolateral	3	9	15
Posterolateral	4	10	-
Inferior	5	11	16
Inferoseptal	6	12	13

Diastolic function is assessed by measuring the Trans-Mitral pressure gradients using Doppler Echocardiography.

Diastolic dysfunction is graded according to the filling pattern into:

Grade I – impaired relaxation with normal filling pressures

Grade II – Pseudonormalised mitral inflow pattern

Grade III – reversible restrictive pattern

Grade IV – irreversible restrictive pattern

The systolic and diastolic dysfunction assessed by the above methods is correlated with other variables such as Age, Sex, Smoking, Type of Myocardial Infarction, Killip class.

Killip Classification:

- Class I – no signs of heart failure,
- Class II – crackles in lower lung fields and S3,
- Class III – acute pulmonary edema,
- Class IV – cardiogenic shock).

The patients were clinically monitored for the development of early in-hospital congestive cardiac failure during the period of admission. Patients with Killip class ≥ II were defined as having heart failure. The Killip class is assessed every day and the highest class is taken for consideration. According to Killip classification, the patients were divided into two groups: those without CHF (Killip class = I) and those with CHF (Killip class ≥ II). Other adverse events during in-hospital evolution that could also be related to other factors and not related to LV dysfunction alone, like recurrent angina or early malignant arrhythmias due to electrical instability that could lead to different results were not considered.

Patients were observed during daily in-hospital evolution, after receiving conventional clinical therapy (with betablockers and angiotensin converting enzyme inhibitors). All patients received reperfusion therapy by streptokinase as per standard guidelines.

INCLUSION CRITERIA

- Patients of both sex, aged between 30 and 60 with Acute Myocardial Infarction (STEMI) who are admitted in the Intensive Coronary Care Unit.
- Patients undergoing thrombolysis using streptokinase.

EXCLUSION CRITERIA

- Patients with Non ST elevation myocardial infarction.
- Patients who have contraindications for thrombolysis.
- Patients with previous history of myocardial infarction.
- Patients with complete heart block.
- Patients with atrial fibrillation.
- Patients with other co-morbidities such as Chronic Kidney disease, Chronic Obstructive Pulmonary Disease.

- Patients with prior history of heart failure symptoms.
- Patients with valvular heart disease.
- Patients with cardiomyopathies.

Statistical analysis

Data were represented as mean ± SD or percentage of the total, unless otherwise specified. Statistical analysis was done using SPSS ver. 20. Comparison between continuous variables was done using Mann Whitney U test or ANOVA. ROC curves were plotted to determine the ideal cutoff for Echocardiographic variables for predicting heart failure. Univariate logistic regression was used to compare the clinical and echocardiographic variables with heart failure. The significant variables in univariate analysis were added to a complete model of multivariate logistic regression. P value of <0.05 was considered significant.

OBSERVATIONS AND DATA ANALYSIS

All the 50 patients included in the study presented with isolated acute ST elevation Myocardial Infarction. All the patients had regional wall motion abnormalities in their Echocardiogram and underwent thrombolysis. The study group included 36 males and 14 females. The difference in sex wise distribution is obvious, as only patients between the age of 30 and 60 were included in the study and in this age group STEMI is more common in males.

The age wise distribution chart shows that the incidence of STEMI increases as the age advances. It is also seen that the maximum number of female patients are in the 56-60 group, implying that the risk for MI increases during the post menopausal period.

Table 1 - AGE GROUP and SEX wise distribution

		SEX		Total
		Female	Male	
AGE GROUP	<40	1	3	4
	41-45	2	4	6
	46-50	3	8	11
	51-55	2	11	13
	56-60	6	10	16
Total		14	36	50

Table 2 - Distribution based on type of MI					
MI type	**AGE**	**SEX**		**SBP**	**DBP**
	Mean ± SD	**Female**	**Male**	**Mean ± SD**	**Mean ± SD**
		Count	**Count**		
Inferior	52 ± 7	7	11	117 ± 30	83 ± 20
Anterior	51 ± 6	7	25	133 ± 26	89 ± 19
p (ANOVA)	0.426	0.325[f]		0.057	0.303

Table 2 compares the MI location with age, sex and admission blood pressure. There was no significant difference in age or sex wise distribution between the two groups mean age in the Anterior MI group was 51 years compared to a mean age of 52 in the Inferior MI group. Males had higher incidence of Anterior MI compared to females in whom the incidence of anterior and inferior was same. However the difference was not statistically significant when compare with a fisher exact test ($p=0.325^{f}$). The mean systolic blood pressure was higher in the Anterior MI group (133 ± 26 vs 117 ± 30) however the difference did not reach statistical significance ($p>0.05$). The diastolic blood pressure was not much different (83mmHgcompared with 89mmHg) in both the groups ($p>0.05$).

Table 3 – Echocardiographic parameters in MI types				
	Type of Myocardial infarction			
	Inferior	**Anterior**	Comparison between groups (ANOVA)	
	Mean ± SD	Mean ± SD	F	p
LVEF	48 ± 13	42 ± 10	4.281	0.044*
LVEDD	4.5833 ± 0.8847	4.7750 ± 0.8116	0.602	0.441
LVESD	3.4278 ± 0.9067	3.7781 ± 0.8003	2.006	0.163
RWMI	1.3056 ± .0770	1.5742 ± 0.3309	11.421	0.001*

Table 3 shows the various Echocardiographic parameters and their distribution among the MI types. The mean LVEF (Left Ventricular Ejection Fraction) is lower in the Anterior MI group ($p<0.05$) implying that Anterior Myocardial Infarction patients are more likely to develop LV systolic dysfunction, which is not surprising given the fact that Anterior MI tends to affect larger area of left ventricle. Both left ventricular end systolic and end diastolic diameters were higher in Anterior MI patents, but the difference was not statistically significant ($p>0.05$). The regional wall motion scoring index (RWMI) was higher in Anterior MI patients (1.57 ± 0.33 vs 1.30 ± 0.07). This

was statistically significant ($p<0.05$) and can be explained by the difference in infarct dimensions among the groups.

Table 4 – Diastolic dysfunction in MI types			
Grading of diastolic dysfunction	**MI type**		
	Inferior	**Anterior**	**Total**
Normal	8	18	26
Grade 1	8	12	20
Grade 2	2	2	4
Grade 3	-	-	-
Grade 4	-	-	-
p (fisher's exact)	0.606		

Table 4 shows the distribution of diastolic dysfunction between the MI groups. There was no significant difference in the distribution of diastolic dysfunction among the MI types ($p>0.05$)**.** Diastolic dysfunction tends to be equally distributed between anterior and inferior myocardial infarction groups. 26 patients (52%) had normal LV filling, and 24 patients (48%) had diastolic dysfunction. Among them 20 had grade I diastolic dysfunction and only 4 had grade II diastolic dysfunction. None had grade III or grade IV diastolic dysfunction which is uncommon in the setting of first AMI.

Table 5 – Systolic and Diastolic dysfunction in MI types						
MI type	**LVEF**		**RWMI**		**DD**	
	≤40%	>40%	<1.7	≥1.7	Absent	Present
Inferior	5	13	18	0	8	10
Anterior	20	12	21	11	18	14
p (fisher's exact)	0.038*		0.004*		0.557	

Table 6 – Systolic and Diastolic dysfunction in males and females						
Sex	**LVEF**		**RWMI**		**DD**	
	≤40%	>40%	<1.7	≥1.7	Absent	Present
Female	4	10	12	2	10	4
Male	21	15	27	9	16	20
p (fisher's exact)	0.114		0.481		0.119	

Table 5 shows the distribution of abnormal echocardiographic indices of systolic and diastolic dysfunction, between the MI types. The left ventricular Ejection Fraction was significantly less in the anterior MI group compared to the inferior MI group ($p<0.05$). Similarly the regional wall motion scoring index also was higher in the anterior MI group ($p<0.05$). However Diastolic dysfunction was equally distributed between the MI types.

Table 6 shows the sex wise distribution of echocardiographic indices. There was no significant difference between the sexes.

Table 7 – Systolic and Diastolic dysfunction in different age groups						
Age group	**LVEF**		**RWMI**		**DD**	
	≤40%	>40%	<1.7	≥1.7	Absent	Present
<40	0	4	4	0	3	1
41-45	3	3	3	3	2	4
46-50	9	2	10	1	5	6
51-55	7	6	8	5	6	7
56-60	6	10	14	2	10	6
p (fisher's exact)	0.046*		0.116		0.619	

In table 7, apart from asymmetric distribution of depressed left ventricular ejection fraction among different age groups, the other parameters - RWMI and DD were equally distributed.

Table 8 – Hospital stay for different groups (in days)					
Clinical variable		**ICCU**		**TOTAL HOSP STAY**	
		Mean	**Standard Deviation**	**Mean**	**Standard Deviation**
SEX	**Female**	2.545	0.688	6.636	0.674
	Male	2.676	0.768	6.912	0.996
MI type	**Inferior**	2.750	0.856	7.000	1.155
	Anterior	2.586	0.682	6.759	0.786
DM	**No**	2.703	0.740	6.892	0.966
	Yes	2.375	0.744	6.625	0.744
SM	**No**	2.500	0.673	6.545	0.671
	Yes	2.783	0.795	7.130	1.058
HF	**No**	2.368	0.496	6.684	0.671
	Yes	2.846	0.834	6.962	1.076

Table 8 shows the duration of hospitalisation of patients. There was no difference in number of days of ICCU stay or hospital stay between males and females. Diabetics had higher duration of ICCU and hospital stay compared with non-diabetics. Similarly smokers also had a higher duration of hospitalisation. Patients with symptoms of heart failure had longer duration of ICCU as well as total hospital stay as expected.

Analysis of early in hospital congestive heart failure in AMI patients:

All the 50 patients were monitored for development of early in-hospital congestive heart failure (defined as Killip class ≥ II). The highest class during the hospital stay was considered for analysis.

Table 9 – Heart failure in different MI types		
MI type	**Heart Failure (Killip class ≥ II)**	
	Absent	Present
Inferior	9	9
Anterior	10	22
p (fisher's exact)	0.233	

There was no significant difference in incidence of heart failure among the different MI types ($p>0.05$). Table 9 depicts the figures.

Table 10 – Heart failure in different age groups		
Sex	**Heart Failure (Killip class ≥ II)**	
	Absent	Present
<40	3	1
41-45	3	3
46-50	4	7
51-55	3	10
56-60	6	10
p (fisher's exact)	0.425	

Table 10 shows the incidence of heart failure following AMI in different age groups. Older patients had higher percentage of heart failure symptoms. However the difference in heart failure rates among the age groups, did not reach statistical significance ($p>0.05$).

Table 11 – Heart failure in different sex groups		
Sex	**Heart Failure (Killip class ≥ II)**	
	Absent	Present
Male	11	25
Female	8	6
p (fisher's exact)	0.110	

Table 11 shows the sex wise difference in development of heart failure. Though the number of males developing heart failure was higher the difference was not statistically significant ($p>0.05$).

Table 12 – Baseline Clinical variables in Heart failure groups			
Clinical variables	**Heart Failure (Killip class ≥ II)**		P value
	Absent (n=19)	Present (n=31)	
Age (years)	50.16 ± 6.98	52.29 ± 5.23	0.336
Systemic Hypertension	6 (50%)	6 (50%)	0.496
Admission SBP	136.32 ± 29.10	121.55 ± 26.38	0.143
Admission DBP	90.53 ± 16.82	83.87 ± 20.11	0.347
Diabetes Mellitus	1 (8.3%)	11 (91.7%)	0.018*
Smoking	6 (24%)	19 (74%)	0.079
Hyperlipidemia	4 (33.3%)	8 (66.7%)	0.490
No. of days in ICCU	2.37 ± 0.50	2.85 ± 0.83	0.046*
Total no. of days in hospital	6.68 ± 0.67	6.96 ± 1.08	0.514

The baseline clinical parameters are compared between the heart failure groups in table 12. The mean age of patient with heart failure was 52.29 years compared to 50.16 years for the normal group. This difference was not significant ($p>0.05$). Prior history of systemic hypertension was comparable between the groups. The admission mean systolic blood pressure was 15 mmHg lower (121.55 vs 136.32) in the heart failure group. This difference can be attributed to the patients with cardiogenic shock in the heart failure group. The

difference was not statistically significant (p>0.05). The mean diastolic blood pressure was also marginally higher (90.53 vs 83.87) in the normal group.

Table 13 – Echocardiographic variables in heart Failure groups					
Echocardiographic Variables	**No Heart Failure (n=19)**		**Heart Failure (n=31)**		P value (Mann-Whitney)
	Mean	SD	Mean	SD	
LVEDD	4.28	0.72	4.96	0.80	0.003*
LVESD	3.10	0.69	3.99	0.76	<.001*
LVEF	52.53	9.61	38.81	8.53	<.001*
RWMI	1.38	0.25	1.53	0.31	0.038*

Table 13 depicts the various echocardiographic parameters in the two groups. The mean Left ventricular end diastolic diameter was higher in the heart failure group with mean 4.96 ± 0.8 cm compared to the patients without heart failure who had a mean of 4.28 ± 0.72 cm. The difference was statistically significant when compared using the Mann Whitney u test (p<0.05).

Similarly the LV End Systolic diameter was higher in the heart failure group mean 3.99 ± 0.76 cm compared to normal group mean of 3.10 ± 0.69 cm.

Again the difference was statistically significant with a p <0.05. LV Ejection Fraction the main parameter for assessing systolic function was lower in the heart failure group 38.81 ± 8.53 vs 52.53 ± 9.61. The difference was highly significant statistically (p <0.001). The regional wall motion scoring index also was higher in the heart failure group as expected. The mean in the heart failure group was 1.53 ± 31 compared with the mean of 1.38 ± 0.25 in the normal group. The difference was statistically significant (p<0.05). From this table we can conclude that the echocardiographic parameters are significantly abnormal in the heart failure group compared with the normal group. Among the echocardiographic parameters the LV ejection fraction had the most significant difference between the groups.

Table 14 – Diastolic dysfunction in Heart failure groups

Grading of diastolic dysfunction	**Heart failure**		
	Absent	**Present**	**Total**
Normal	15	11	26
Grade 1	3	17	20
Grade 2	1	3	4
Grade 3	-	-	-
Grade 4	-	-	-
p	0.008*		

Table 14 shows the distribution of diastolic dysfunction between the normal and heart failure groups. 24 out of 50 patients (48%) had diastolic dysfunction as assessed by Doppler Echocardiography. 20 patients with heart failure had diastolic dysfunction, whereas only 4 without heart failure symptoms had diastolic dysfunction by echocardiography. This difference was statistically significant (with $p<0.05$). Out of the 24 patients with Diastolic dysfunction 20 had Grade I diastolic dysfunction and 4 had Grade II diastolic dysfunction. Among the 4 patients with Grade II diastolic dysfunction 3 had signs of heart failure. Hence it appears that diastolic dysfunction as detected by echocardiography has a significant relationship with heart failure.

Table 15 – ROC curves for LVEF and RWMI

Variable	Area under curve (AUC)	Standard error	95% confidence interval	p
LVEF	0.862	0.0539	0.735 to 0.943	<0.0001*
RWMI	0.671	0.0750	0.523 to 0.797	0.0229*

Table 16 – Cut off values based on ROC curves for LVEF and RWMI				
Variable	**Sensitivity**	**Specificity**	**PPV**	**NPV**
LVEF ≤ 40	74.2	89.5	92.0	68.0
RWMI ≥ 1.7	20.5	94.7	86.3	42.2

Table 15 shows the ROC (Receiver Operating Characteristics) curve parameters. The ROC curve was plotted to assess the predictive (diagnostic ability of the Echocardiographic parameters in detecting heart failure).

LV Ejection Fraction had an AUC (area under the curve) of 0.862 which denotes that the LVEF has very high diagnostic accuracy in predicting heart failure (p value of <0.0001).

Wall motion scoring index had an AUC of 0.671 with a p value of <0.05 also had good ability to detect heart failure but not to the extent of LV Ejection Fraction.

In table 16, based on the ROC curves cut offs were estabilished which helps to classify patients accutately. LVEF ≤ 40 had the best predictive ability. Similarly RWMI >1.7 though did not have the highest predictive ability was chosen since it had very good specificity of 90%.

Table 17 – Univariate Regression analysis for determining variables associated with early Heart Failure				
Variable	**Odds ratio**	**Regression coefficient**	**Standard error**	**P**
Age	1.063	0.061	0.050	0.222
Sex	3.030	1.109	0.650	0.088
MI type	2.200	0.788	0.606	0.193
SBP	0.980	-0.020	0.012	0.084
DM	9.900	2.293	1.094	0.036*
SM	3.431	1.233	0.616	0.045*
LVEF ≤ 40	24.437	3.196	0.853	<0.001*
RWMI ≤ 1.7	8.571	2.148	1.097	0.049*
DD	6.818	1.920	0.676	0.005*

Table – 17 shows the univariate regression analysis correlating all the clinical and echocardiographic variables with heart failure. There was no significant correlation between clinical parameters age, sex, MI location, admission Systolic blood pressure. Diabetes was significantly correlating with heart failure. Diabetic patients were 9 times more likely to develop heart failure symptoms following AMI compared with non-diabetics (p value of <0.05). Similarly smokers were also 3 times more likely to develop heart failure compared to non-smokers (p value of <0.05)

Among the Echocardiographic parameters LV Ejection Fraction <40 had the strongest predicting ability with an odds ratio of 24 which was statistically highly significant (p<0.001). Wall motion scoring index and diastolic dysfunction also had good ability to predict heart failure with an odds ratio of 9 and 7 respectively (p value <0.05).

Table 18 – Multivariate Logistic regression				
Variable	**Odds ratio**	**Regression Coefficient**	**Standard error**	**p**
LVEF ≤ 40	14.386	2.666	0.915	0.004*
DD	5.738	1.747	0.829	0.035*
RWMI ≥ 1.7	4.862	1.581	1.514	0.218
Constant	0.212	-1.552	0.608	0.011*

Table 18 shows the Multivariate regression model where all the three significantly correlating echocardiographic parameters (in univariate analysis) are simultaneously analysed for any confounders and interdependence among the variables. The overall model was statistically significant with a p value of <0.001. LV Ejection Fraction and Diastolic Dysfunction correlated significantly with heart failure. The corrected odds ratio for LVEF ≤ 40 for predicting heart

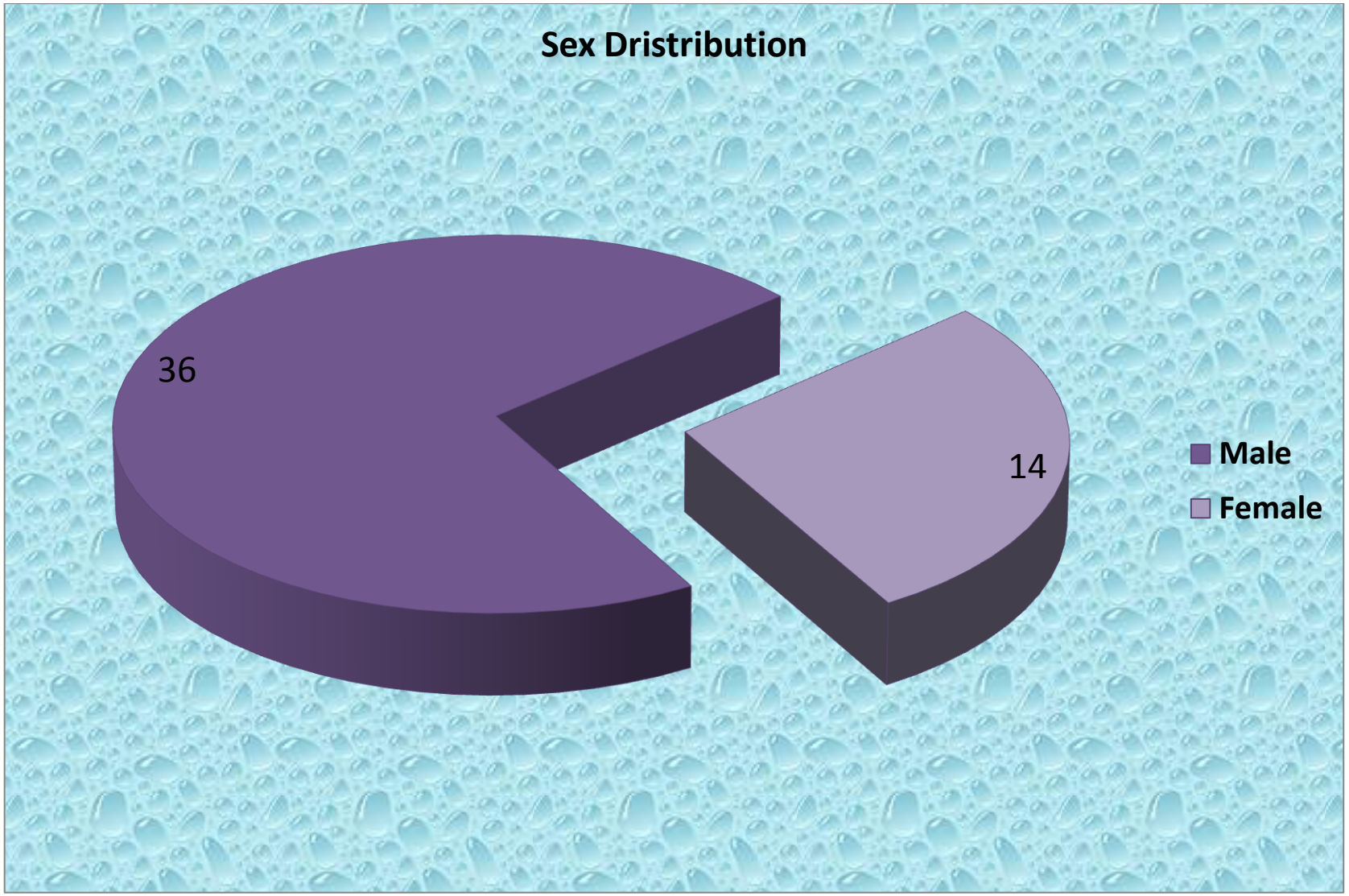
failure was 14.38 ie a patient with a LVEF of less than 40% is 14 times more likely to develop heart failure symptoms than a patient with LVEF >40. The corrected odds ratio for Diastolic Dysfunction was 5.74, and it was statistically significant with a p value of 0.035.

However the Regional Wall Motion Scoring Index though had a higher odds associating it with heart failure, it was not statistically significant within the model. This paradox is likely due to the fact that LV ejection fraction and Wall Motion Scoring are highly inter-dependent variables. When both of them are included in the model it becomes superfluous. Further when we look at the univariate analysis the correlation between LV Ejection Fraction and heart failure is better than that between Regional Wall Motion Scoring Index and heart failure. This could be because of the complex and operator dependent nature of the index.

All the clinical and echocardiographic variables couldn't be analysed in the same Multivariate Logistic Regression model, making it less robust as the sample size was small and the number of variables to sample ratio should be maintained more than at least 1:10 or preferably 1:20. So only the three main echocardiographic variables were included in the regression model.

When backward conditional elimination of variables was followed, Wall motion scoring index was removed from the model and only LV Ejection Fraction and Diastolic Dysfunction remained. On the whole the model was able

to accurately classify the patients into heart failure or normal in 80% of the cases.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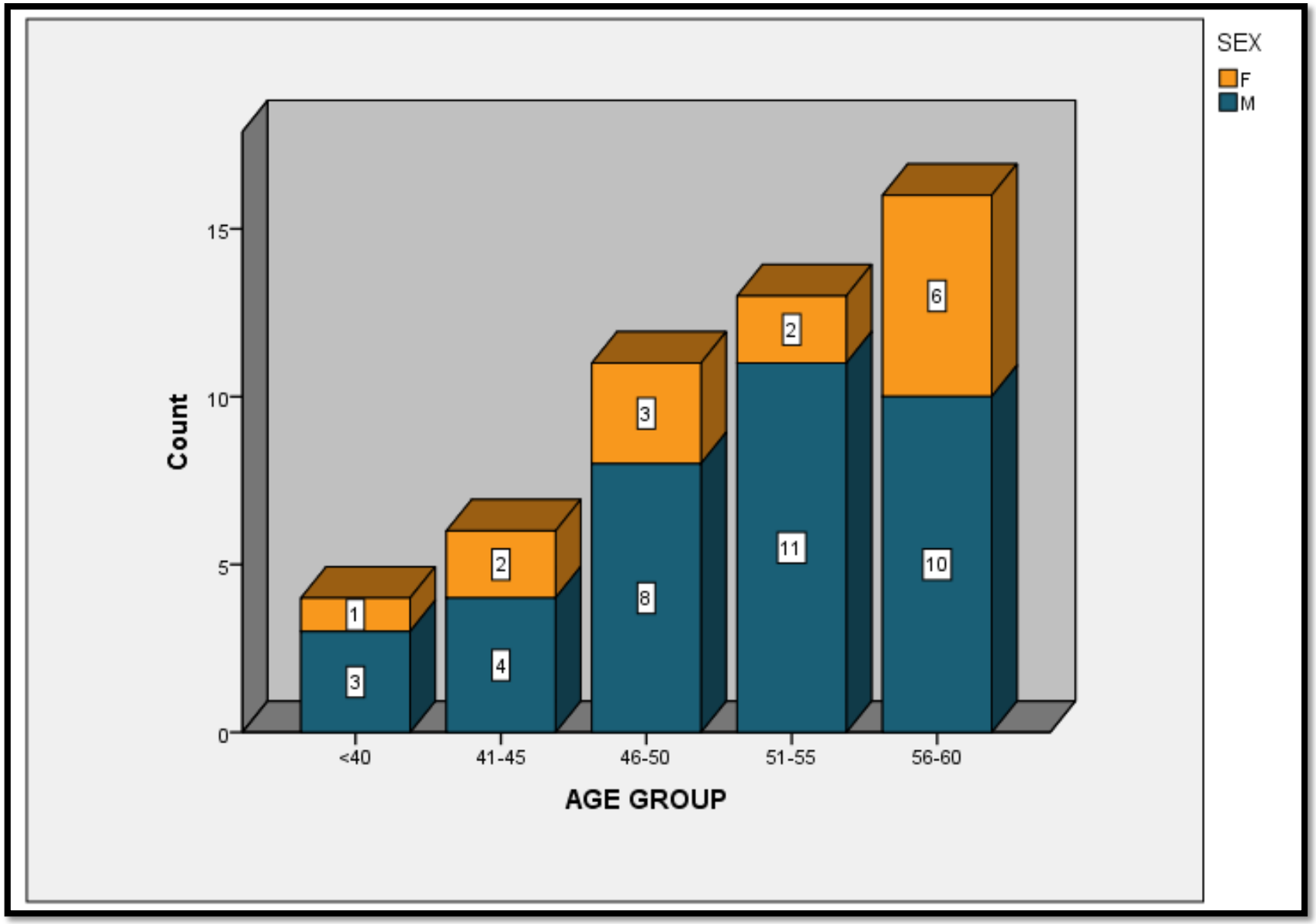
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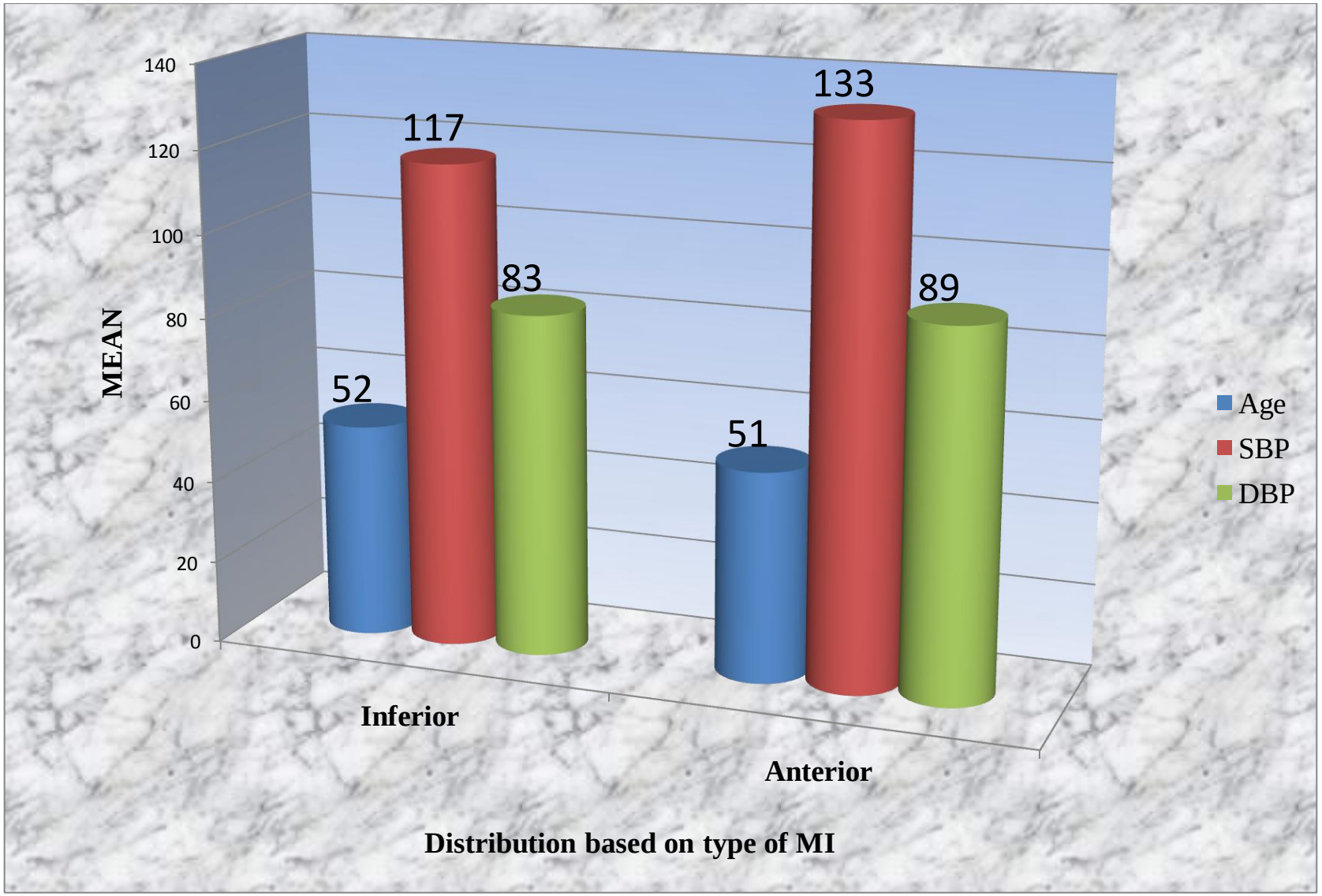
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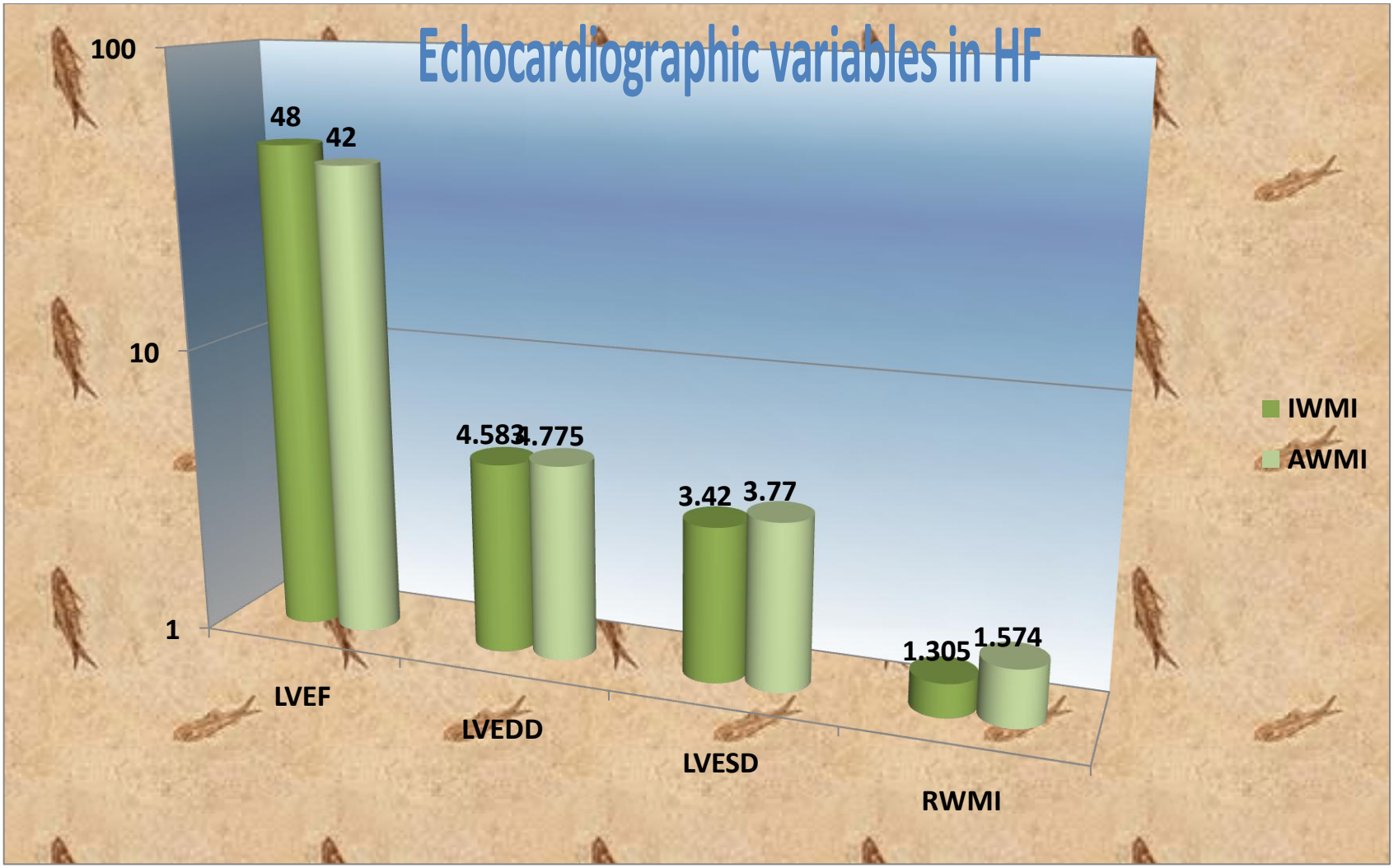
Distribution based on type of MI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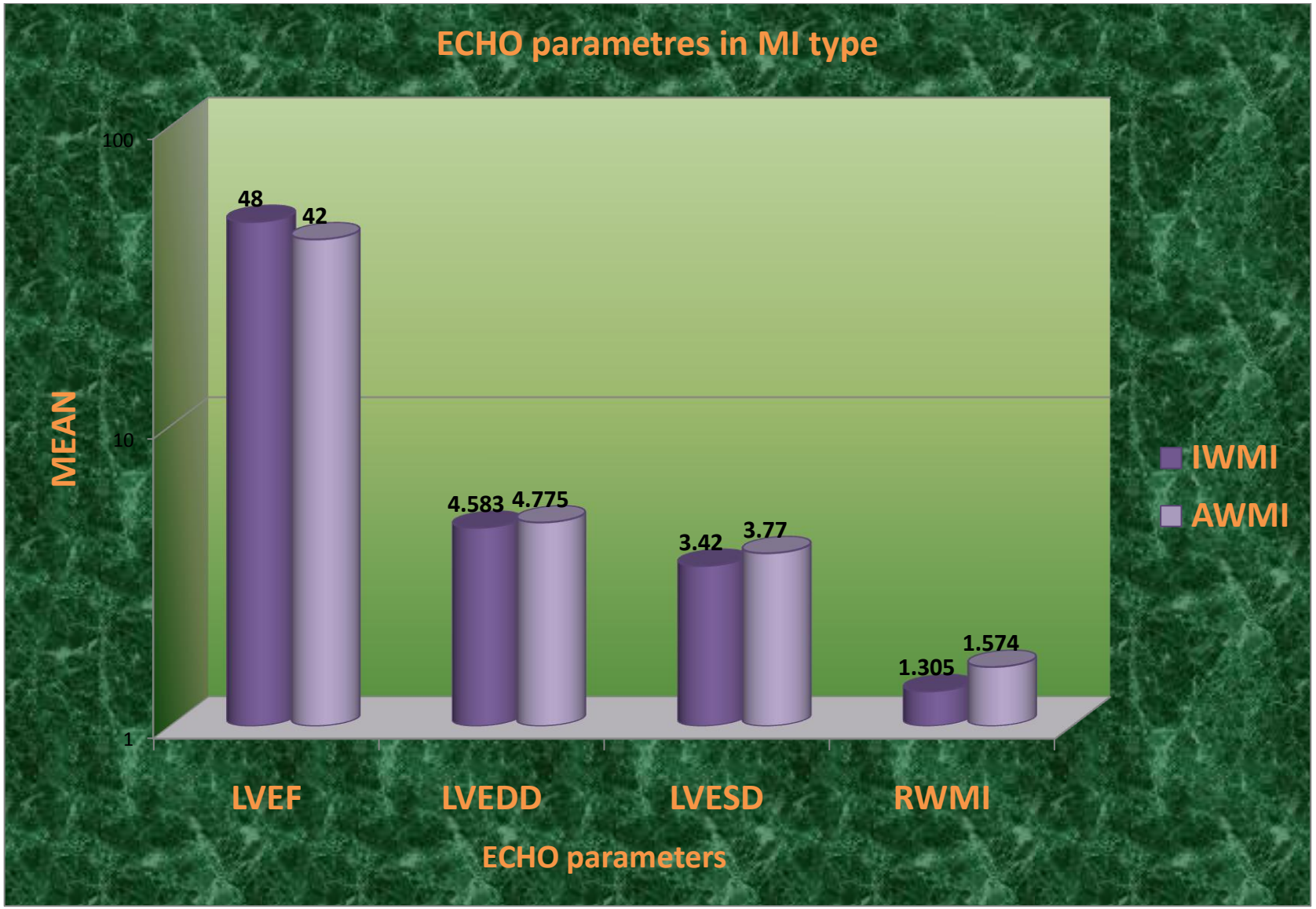
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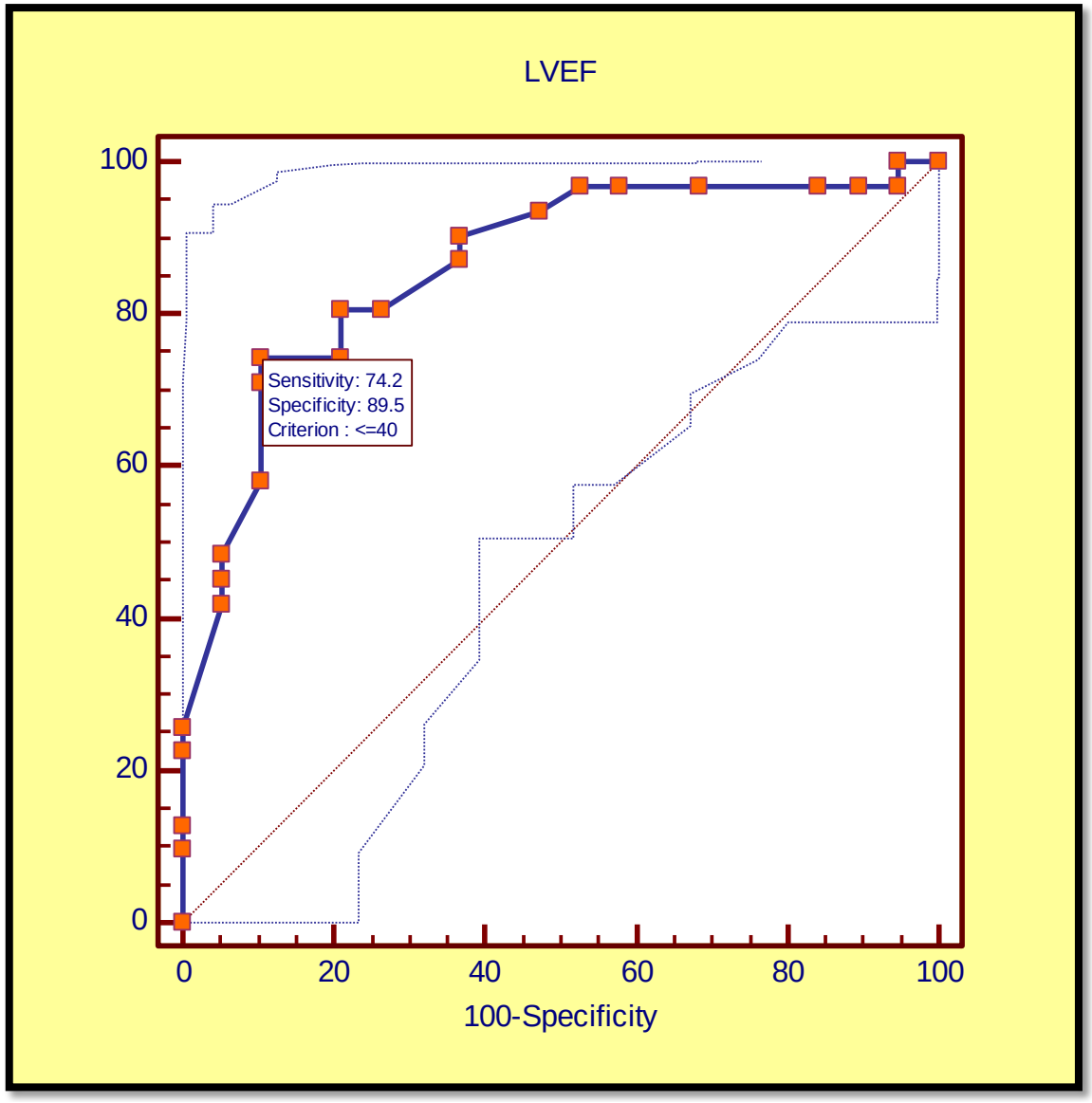
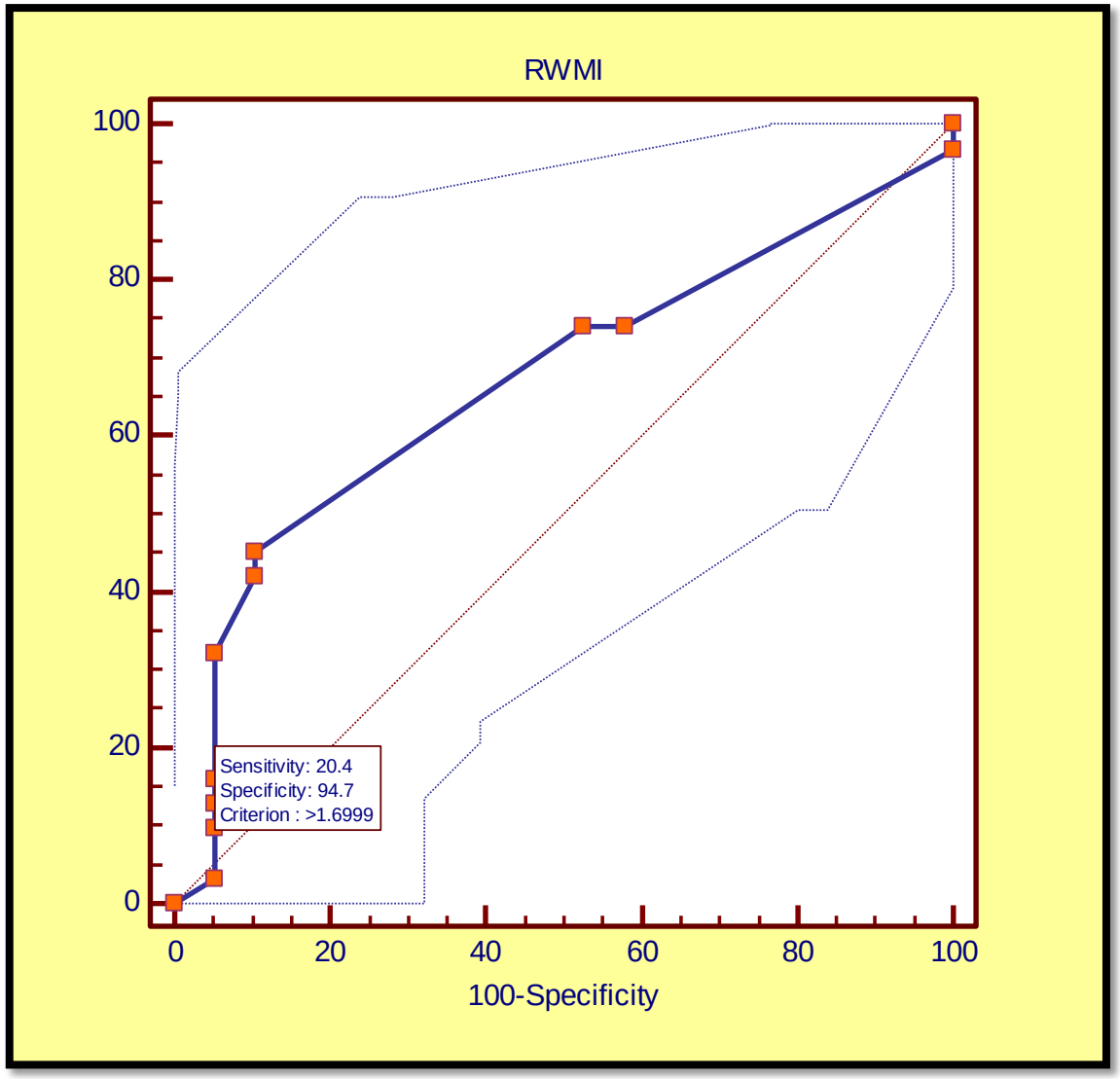
42
4.583
4.775
3.42
3.77
1.305
1.574
IWMI
AWMI
LVEF
LVEDD
LVESD
RWMI
ECHO parameters



ROC curve showing the diagnostic performance of LVEF



ROC curve showing the diagnostic performance of RWMI

DISCUSSION

Fifty patients with first Acute Myocardial Infarction were evaluated in the study. The study mainly focuses on the echocardiographic evaluation of patients with AMI and identification of left ventricular systolic and diastolic dysfunction in them.

In our study 50% (n=25) of the patients had systolic dysfunction (LV ejection fraction <40%). As patients with previous MI, heart failure symptoms or valvular heart disease have been excluded, the systolic dysfunction can be predominantly attributed to the index coronary event. In a similar Portuguese study by PS Mateus et al,[41] 56% had LV systolic dysfunction (they had used LEVF cut-off of 45%). The TRACE trial[42] had a more stringent cut off (LVEF<35%) and found that 40% had Systolic dysfunction. In a Kosovo study by Kocinaj D et al[43] 48% of patients with first AMI had LV systolic dysfunction. Our study results were comparable with other similar studies with similar cut-off for detecting LV systolic dysfunction.

A total of 24 out of 50 patients (48%) had restrictive filling pattern in echocardiogram (diastolic dysfunction). In a similar study by S H Poulsen et al,[45] 35% of the patients had impaired LV filling pattern. However the range varied widely from 20% in a study by Whalley et al[44] to 65% in a study by LP Souza et al.[42] This wide variation among studies can be explained by the fact that different echocardiographic parameters and different criteria were used to

identify patients with diastolic dysfunction. Our study which graded diastolic dysfunction based on the ASE/EAE guidelines[46] pegged the incidence of diastolic dysfunction at 48% following AMI, which was midway between the extreme ranges reported in other studies.

The age, sex wise distribution of systolic and diastolic dysfunction was not statistically significant. Systolic dysfunction was found more frequently in patients with Anterior MI compared with inferior MI. Many studies in the past had similar results comparing the site of infarction with LV ejection fraction. In the study by Mc Clements BM et al,[47] the LV ejection fraction was 8% lower for anterior compared with inferior MI.

The Regional wall motion scoring index was significantly higher in the Anterior MI group. The same has been shown in other similar studies by McClements BM et al, [47] Souza et al.[42]

The incidence of heart failure symptoms (as defined by Killip class ≥ II) was 63% (n=31). This was both at the time of hospital admission as well as during the subsequent stay in the hospital. In the Souza et al[42] study the incidence of heart failure was 44%. In the study of Yuasa[51] et al which included CHF among other end points such as paroxysmal atrial fibrillation, ventricular tachycardia, ventricular fibrillation, AV block, pericardial effusion and cardiac rupture, the prevalence of CHF was only 19%. The Valsartan in

Acute Myocardial Infarction trial (VALIANT) trial[48] showed an incidence of 23.1 at admission, with a higher number being discharged with a diuretic.

In our study only LVEF and presence of DD were strong independent predictors for the development of early CHF following AMI. In the Souza et al[42] study only LVEF (<45%) was a statistically significant echocardiographic index for predicting early CHF. Wall motion scoring index, when adjusted for age also was a good predictor. However in our study wall motion scoring index though correlated well in the univariate analysis did not reach statistical significance in the multivariate regression analysis. In the above study they also correlated Myocardial Performance Index (MPI) with heart failure. However even though MPI is a global myocardial performance index including both systolic and diastolic echocardiographic indices, it did not significantly correlate with development of heart failure symptoms.

Lavine[49] in a retrospective study also found that LVEF was superior to other indices in predicting development of heart failure in patients without any clinical evidence of CHF at admission during the first 15 days of admission following AMI. In the study by schwammenthal et al.[50] also reported a LVEF ≤ 0.40 as a powerful and independent predictor of poor outcome

Regional wall motion scoring index of LV systolic function did not provide any additional prognostic information over LV ejection fraction. This paradox may be explained by the fact the both variables are interdependent and

Wall motion analysis is a complex and highly subjective quantification index with a high degree inter-observer variation. In comparison LVEF is an easily measurable echocardiographic index and is easily reproducible, thereby the errors in LVEF estimation are vey less. Similar findings were obtained in studies by Yuvasa et al [51] and DeMichele. [52]

Similar to many other studies like Souza et al, Diabetes and Smoking were predictors of early in hospital heart failure in the univariate analysis. Further studies are needed with larger study population to assess the interdependence of clinical and echocardiographic parameters.

Limitations of the study

Though extremes of age were excluded in the study to minimise the influence of age on diastolic dysfunction, the study group included a sizeable number of diabetics and hypertensives who might have pre-existing diastolic dysfunction, which might confound the results.

Our results about the prognostic influence of Regional Wall Motion scoring Index for first ST-elevation AMI was inconclusive, due to the limited number of patients in the study group and operator dependant nature of the index.

We couldn’t analyse all the clinical and echocardiographic variables simultaneously in the multivariate logistic regression as the study group was small and the variable to population ratio (1:10) would exceed the guidelines.

The recent global indices of systolic and diastolic function like the MPI were not included for analysis.

The individual diastolic parameters such as E/A ration, DT were not compared with heart failure.

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CONCLUSIONS

- ✓ In this study population of 50 patients with first AMI and without previous heart failure symptoms, the incidence of early in-hospital congestive heart failure was 62% (n=31).
- ✓ 48% of the patients with first AMI had diastolic dysfunction.
- ✓ 50% of patients had systolic dysfunction (defined as ejection fraction of ≤ 40%).
- ✓ 22% of patients had a Regional Wall Motion Scoring Index of ≥1.7.
- ✓ Left ventricular ejection fraction was the most important predictor of early heart failure (p=0.004).
- ✓ The cut off ≤ 40% of LVEF had a high predictive value (74% sensitivity 90% specificity) in detecting patients who are likely to develop heart failure symptoms.
- ✓ Presence of Diastolic dysfunction also predicted early heart failure following AMI accurately (p=0.035).
- ✓ Regional Wall Motion Index was inferior compared to LVEF and presence of DD in predicting heart failure (p=0.218).
- ✓ Smokers were more likely to develop early in-hospital heart failure following AMI (p=0.045).
- ✓ Diabetes was one of the important risk factors for the development of heart failure following AMI (p=0.036).

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INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Echocardiographic evaluation of left ventricular systolic and diastolic function in patients with acute myocardial infarction

Principal Investigator : Dr.J..Stalin Roy, PG in MD(GM)

Designation : PG in MD(GM)

Department : Department of Medicine
Government Stanley Medical College,
Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 18.04.2011 at the Modernized Seminar Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate form the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

PROFORMA

S.no:

Date:

Name:

Age:

Sex:

Address:

Contact phone no:

Occupation:

Past history of Diabetes/ Hypertension:

Family history of Coronary Artery Disease:

Presenting complaints:

Pulse: BP:

CVS: RS:

Killip Class:

ECG (Type of Myocardial Infarction, Probable site of coronary occlusion):

Routine investigations:

Random blood sugar:

Serum Creatinine in mg/dl:

Blood urea nitrogen in mg/dl:

ECHOCARDIOGRAPHY

Left Ventricular Ejection Fraction:

Regional Wall motion index:

Diastolic dysfunction grade:

Clinical monitoring for Cardiac Failure during hospital stay							
	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
JVP							
S3, S4							
Basal Crackles							
Pedal Edema							

NO.	NAME	IP NO	AGE	SEX	TYPE OF MI	SBP	DBP	DM	SHT	SM	HL	KILLIP	ICCU	HOSP	A/D	LVEDD	LVESD	LVEF	RWMI	DD
1	Noorulla	28378	39	M	ASMI	110	70					1	2	6	A	4.1	2.9	50	1.3125	
2	Anthony	28504	58	M	IPWMI/RVMI	120	70		+	+		1	2	8	A	4.7	3.1	60	1.25	
3	Arunachalam	28778	57	M	IPWMI/RVMI	160	100				+	1	3	6	A	5.6	4.2	48	1.25	1
4	Durgammal	31303	59	F	IPWMI/RVMI	82	56	+			+	4			D	5.8	4.9	29	1.375	1
5	Periyanthevan	31772	42	M	AWMI	150	90	+		+		2	2	8	A	6.6	5.3	38	1.75	1
6	Rathinam	31787	50	M	IWMI/RVMI	84	60			+		4	5	10	A	5.1	4.2	32	1.25	
7	Anthony	32621	54	M	AWMI	140	100			+		1	3	7	A	3.4	2	68	1.25	
8	Rani	32954	50	F	ALMI	200	110		+			1	3	6	A	4	3	48	1.375	
9	Sivaraj	33002	52	M	AWMI	150	100	+	+			1	4	6	A	5.1	4.2	35	1.375	
10	Parvathy	33062	45	F	IPWMI	210	140		+			1	2	7	A	3.4	2.2	64	1.375	
11	Kasi	33159	52	M	ASMI	130	90			+	+	2	3	8	A	4.3	3.4	37	1.25	
12	Jayaraj	33215	43	M	AWMI	120	100			+		1	4	8	A	5	4.2	33	1.875	
13	Sundar	33446	50	M	IWMI/RVMI	96	64	+		+		3			D	5.1	4.3	32	1.25	2
14	Dhanalakshmi	33617	52	F	AWMI	130	80	+	+			1	2	7	A	5.3	3.9	53	1.375	
15	Palani	33622	50	M	ASMI	120	70					1	3	6	A	5	4	39	1.25	
16	Mohamed Hasen	33682	58	M	IPWMI	110	70			+		1	3	7	A	4.7	2.9	69	1.25	
17	Kamalakannnan	33702	58	M	ASMI	150	100			+	+	1	3	6	A	4.8	3.6	48	1.5625	
18	Lakshmi	33773	59	F	IPWMI	120	90				+	1	3	6	A	4.4	2.9	60	1.375	
19	Karpagam	33791	40	F	IPWMI	100	70					1	3	6	A	5.1	3.7	52	1.375	
20	Rajammal	33960	45	F	IWMI	120	100					1	3	7	A	4	2.8	58	1.25	
21	Munuswamy	34821	58	M	ASMI	140	100				+	2	2	6	A	3.4	2.9	35	1.75	
22	Aruldas	34883	54	M	AWMI	130	90	+		+		1	3	6	A	5	4	39	1.375	1
23	Manoharan	35019	53	M	IWMI	120	90			+		2	3	8	A	5.6	4.2	44	1.125	1
24	Mohanlal	35167	46	M	ASMI	130	90		+			1	2	6	A	4.7	3.9	35	1.25	
25	Thanikachalam	35342	59	M	ASMI	120	90	+			+	2	2	7	A	5	4	40	1.5625	

SBP –Systolic blood pressure, DBP – Diastolic blood pressure, DM – Diabetes, SHT – Systemic hypertension, SM – Smoking, HL – Hyperlipidemia, ICCU – No of days in ICCU, HOSP – total no of days in hospital, A/D – Alive/Dead, LVEDD Left Ventricular End Diastolic Dimension, LVESD – Left Ventricular End Systolic Dimension, LVEF – Left Ventricular Ejection Fraction, RWMI – Regional Wall Motion Scoring Index, DD – Diastolic Dysfunction (grading).

NO.	NAME	IP NO	AGE	SEX	TYPE OF MI	SBP	DBP	DM	SHT	SM	HL	KILLIP	ICCU	HOSP	A/D	LVEDD	LVESD	LVEF	RWMI	DD
26	Rajammal	35513	48	F	ASMI	130	90		+			3	4	7	A	6.5	5.2	38	1.5625	
27	Krishnamorthy	35549	53	M	IPWMI	100	80					1	2	6	A	4	3.2	42	1.375	
28	Rajammal	35718	59	F	AWMI	130	90				+	1	2	6	A	3.8	2.4	66	1.25	
29	Saraswathi	35746	57	F	IWMI	140	100				+	1	2	8	A	3.2	2.1	60	1.25	
30	Puspharaj	35821	51	M	ASMI	140	100			+		3	3	8	A	4.8	4	32	2.125	1
31	Venkatesan	35881	40	M	ALMI	130	80			+		1	2	7	A	4.1	2.8	58	1.25	1
32	Pasupathy	35916	52	M	ASMI	140	100		+	+		2	2	6	A	5.5	4.8	28	1.75	
33	Lakshmi	35922	50	F	AWMI	130	90					1	2	7	A	4	3.2	42	1.375	
34	Arumugam	36054	38	M	IPWMI/RVMI	110	80			+		1	3	6	A	3.1	2.2	52	1.375	
35	Dhanalakshmi	36949	58	F	ASMI	82	40	+			+	4			D	5.5	4.8	28	1.75	
36	Eshvaran	37208	50	M	AWMI	160	100		+			1	2	7	A	5.6	4.6	38	1.375	
37	Bharathi	37367	55	F	ASMI	86	50		+			4			D	3.4	2.9	35	1.75	
38	Anbalagan	37442	52	M	AWMI	200	140		+			2	3	8	A	4.2	3.2	44	2.125	2
39	Salavudeen	37483	50	M	IWMI	86	68		+	+		4	4	8	A	5.8	5	28	1.375	1
40	Asaithambi	37520	58	M	ASMI	88	60			+	+	4	3	8	A	5.2	4	48	1.375	
41	Dinakar	37533	56	M	IPWMI	120	80			+		2	2	7	A	3.5	2.6	49	1.375	1
42	Mohan	37542	45	M	ASMI	104	62	+		+		3			D	5.2	4.3	35	1.375	1
43	Muniammal	37683	59	F	IPWMI	120	80					1	2	6	A	4.6	3.4	50	1.25	1
44	Ganesh	37694	53	M	ASMI	130	80			+		2	2	7	A	4.8	3.7	45	1.5625	2
45	Nagalingam	37788	48	M	ASMI	120	90	+		+		1	2	6	A	4.6	3.7	39	1.25	1
46	Perumal	37795	56	M	IPWMI/RVMI	110	90	+		+	+	1	2	6	A	4.8	3.8	39	1.375	1
47	Varadhan	37807	55	M	AWMI	150	100	+				2	2	7	A	5.8	4.7	36	1.9375	2
48	Gopinathan	38743	57	M	AWMI	130	90			+		3	3	6	A	4.9	4	35	1.5	1
49	Kuppuswamy	38900	47	M	ASMI	130	90			+		2	2	6	A	5.3	4.4	38	2.375	
50	Nagabooshnam	38908	44	M	ASMI	150	100			+		1	3	7	A	3.9	2.9	50	2.375	

SBP –Systolic blood pressure, DBP – Diastolic blood pressure, DM – Diabetes, SHT – Systemic hypertension, SM – Smoking, HL – Hyperlipidemia, ICCU – No of days in ICCU, HOSP – total no of days in hospital, A/D – Alive/Dead, LVEDD Left Ventricular End Diastolic Dimension, LVESD – Left Ventricular End Systolic Dimension, LVEF – Left Ventricular Ejection Fraction, RWMI – Regional Wall Motion Scoring Index, DD – Diastolic Dysfunction (grading).